PSEUDOMEMBRANOUS INFLAMMATION OF THE MUCOUS MEMBRANES CAUSED BY THE PNEUMOCOCCUS.*

REVIEW OF THE LITERATURE AND REPORT OF A CASE OF PNEUMOCOCCIC PSEUDOMEMBRANOUS EXUDATION ON THE MUCOUS MEMBRANES OF THE MOUTH, TONGUE, THROAT, NOSE, EYES, GLANS PENIS, ANUS, ETC., COMPLICATING ACUTE LOBAR PNEUMONIA.

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The case here reported is remarkable in one respect only, *i. e.*, the occurrence, during the course of an attack of acute lobar pneumonia in a boy of eleven years, of a profuse pseudomembranous exudation upon nearly all the mucous surfaces of the body open to inspection, caused by the pneumococcus (diplococcus pneumoniæ). In addition to the visible fibrinous exudate upon the mucous membranes exposed to view, the case also showed signs of extensive adhesive pleuritis and marked and continued tympanites, with the passage of membranous shreds in the stools, leading to the belief that the fibrinous process involved also the pleuræ and the gastro-intestinal tract. Aside from these features the case showed nothing remarkable, pursuing a severe and protracted course which terminated by gradual lysis with recovery. The case will, therefore, be reported chiefly with reference to the unusual complications, omitting unimportant details relating to the more usual features of the disease—from the clinical side by Cary, who had charge of the case throughout, and from the pathological side by Lyon, who saw the case for the first time on January 17th in consultation with Cary—as follows:

Summary of case: Acute lobar pneumonia of both bases, with development during the first ten days of a profuse pseudomembranous exudate, first upon the tonsils and quickly extending to the mucous membranes of the lips, tongue, mouth, palate, throat, and nose, and transferred to the eyes, glans penis, and anus; physical signs of fibrinous pleuritis; marked and persistent tympanites; membranous shreds and mucus in the stools. Microscopical examination: bacillus diphtheriæ, streptococcus, saccharomyces albicans, etc., absent; diplococcus pneumoniæ present in abundance in the sputum and in the exudate from all locations, grown in cultures and injected into a rabbit and recovered from the organs and blood after death. Diagnosis: pneumococcic infection resulting in acute pneumonia, pleurisy,

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and fibrinous exudation upon the mucous membranes.—T. R., aged eleven years, a boy somewhat undersized and undeveloped for his age, of the best social condition and surroundings, was taken ill on January 5, 1901.

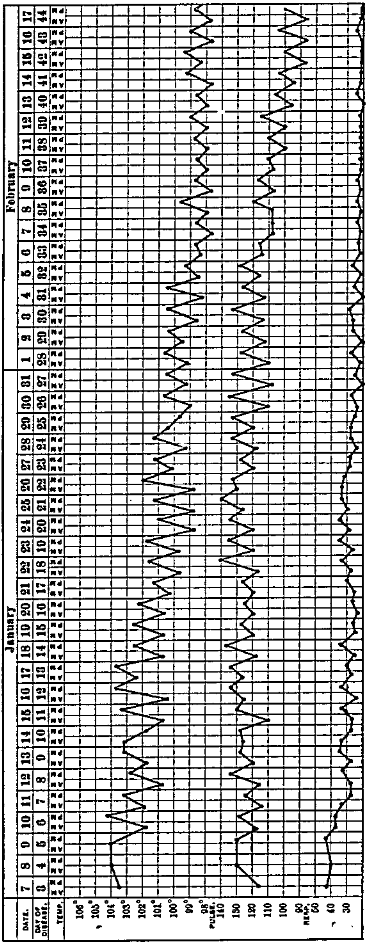
Family History. Negative.

Personal History. The patient has had the usual diseases of childhood. He had been a mouth-breather from enlarged tonsils, which were excised five years ago, since which he has been subject to frequent and severe attacks of follicular tonsillitis, of which as many as eight or nine sometimes occurred during a single season. Throat cultures made during these attacks always proved negative for the diphtheria bacillus. During the past year only a single attack of tonsillitis occurred. One year ago he had a short, slight attack of catarrhal pneumonia, from which he recovered without incident.

Present Illness. The present illness began on January 5, 1901, with sore-throat and fever, which were supposed by the boy's mother to indicate the onset of one of his accustomed attacks of tonsillitis, and he was treated with the usual remedies. On January 7th the family physician, Dr. Cary, was called, and found a reddened sore-throat (without exudation), signs indicating early involvement of the base of the left lung, and temperature 103.5° F., pulse 114, and respirations 42. Next day the temperature was 104° F., pulse 130, respirations 40, and the signs of pneumonia at the left base were more marked. On January 11th signs of consolidation of the right base also appeared, and the patient complained of a painful sore-throat. Examination showed an abundant white exudate covering both tonsils, from which a culture was made and was reported negative for the diphtheria bacillus by Dr. Thomas B. Carpenter, bacteriologist of the City Board of Health. In the meantime a marked vesicular eruption had appeared over the chest, following directly the application of turpentine and oil (1:6). The vesicles were numerous, discrete, and reached the size of a five-cent piece in some instances, and the sores left by them were an annoying feature in the early part of the illness.

On January 12th a herpetic eruption appeared upon the lips; the exudation was more or less general throughout the mouth and throat, and the eyes were the seat of a beginning conjunctivitis. Dr. H. Y. Grant, oculist, was placed in charge of the eyes, and active treatment was directed to them. On January 13th there was marked fibrinous conjunctivitis, and subconjunctival hemorrhages were scattered over the eyes, the lids were swollen and adherent, and photophobia was complained of. The mucous membranes covering the lips, gums, cheeks, margin and under surface of the tongue, hard and soft palate, fauces, tonsils, pharynx (as far back as could be seen), and nose were covered with a continuous, white, adherent exudate, which could be torn off in shreds and pieces, sometimes as large as a twenty-five-cent piece, leaving behind a raw, granular, bleeding surface. The boy could not be restrained entirely from picking at his nose and lips, and his fingers were seen to be bloodstained. He thus apparently transplanted the infection from one place to another.

On the evening of January 15th the glans penis (circumcised) became involved in the same membranous process, and next day was covered in its greater part by a thick, raised, adherent membrane, which extended to the meatus and caused the lips to be glued together, producing difficulty in micturition by pressure. The process did not, however,


DATE.
DAY OF DISEASE.
TEMP.
January
February
7 8 9 10 11 12 13 14 15 16 17 18 19 20 21 22 23 24 25 26 27 28 29 30 31
1 2 3 4 5 6 7 8 9 10 11 12 13 14 15 16 17
3 4 5 6 7 8 9 10 11 12 13 14 15 16 17 18 19 20 21 22 23 24 25 26 27 28 29 30 31 32 33 34 35 36 37 38 39 40 41 42 43 44
M E
106°
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98°
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140
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110
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90
RESP.
50
40
30

extend into the urethra. The membrane could be torn off in pieces, leaving behind a raw bleeding base similar to that seen in the mouth after removal of the exudate.

At this time a large piece of the membrane obtained from the mouth was submitted to Dr. Herbert D. Pease, bacteriologist to the New York State Pathological Laboratory of the University of Buffalo, who reported negatively for the diphtheria bacillus, but noted the presence of frequent diplococci, the nature of which, however, was undetermined, as the examination was somewhat cursory.

Next day, January 16th, an examination showed the anus also involved in a membranous deposit, and the nurse first noted the presence of membranous shreds in the stools. The deposit on the anus extended within the sphincter, though to what distance was not determined, as a speculum could not be introduced without much pain. A shred about two inches long was removed through the anus by winding it upon a match. For several days the abdomen had been distended with marked tympanites. The stools had been rather frequent, soft, contained considerable mucus, and much gas was expelled. The spleen was not palpable, and no rose spots appeared.

At this time the disease was at its height. Both lower lobes were consolidated. The membranous exudate was profuse and showed no signs of yielding to active local treatment. The breathing was noisy and labored, with seeming suffocation from the accumulation of mucus in the upper air passages. The expectoration, which was mostly swallowed, was thick, viscid, mucopurulent. The cough was constant, deep, and gagging. About this time, also, signs of extensive fibrinous pleuritis became marked, chiefly over the left base; the right base was also involved, but not so frankly. The pleural friction over the left base could be felt with the hand on the chest, as well as heard with the stethoscope. The patient's condition seemed critical.

On January 17th the case was first seen by Dr. Lyon in consultation with Dr. Cary, when the disease was at its height, and the following examinations were made by him:

Blood. Leucocytes, 16,600; hæmoglobin, 70 per cent. Differential leucocyte count: neutrophiles, 81 per cent.; small lymphocytes, 12 per cent.; large lymphocytes, 6.5 per cent.; eosinophiles, 0.5 per cent.; 400 counted. Red corpuscles well formed; an occasional normoblast. Fibrin increased. Widal test negative.

Sputa. Mucopurulent, yellowish-white in color, with an occasional reddish streak. Microscopically the sputa showed numerous pus cells, epithelial cells, sometimes containing brown granules (blood detritus), mucus, and fibrin; occasional red blood-corpuscles; a few very small fibrinous casts of the finer tubules with dendritic processes attached. Stained specimens showed numerous large diplococci, with wide capsules, morphologically identical with the diplococcus pneumoniæ. This organism stained by Gram, but the capsule was not clearly stained by Welch's method.

Membranous Exudate from Lips. Thick, white, gelatinous material, which was partly soluble in water, which latter gave the chemical reactions for mucin. The undissolved residue showed typical fibrin filaments and stained by Weigert's method. Stained specimens showed in abundance the same diplococci as found in the sputa, as well as an occasional single coccus. Numerous epithelial cells and leucocytes and much detritus were also found in the exudate. The *diphtheria bacillus, streptococcus, saccharomyces albicans, etc., absent.*

Membranous Exudate from Nose, Glans Penis, and Anus. All of these specimens were similar grossly and microscopically to the pseudomembrane from the mouth, all containing the same encapsulated diplococcus in large numbers, practically in pure culture, except the membrane from the anus, which showed, in addition to the diplococcus, contamination with the usual fecal flora. [It is worthy of note that in the case of the glans penis the exudate was not deposited upon a true mucous membrane, as was the case in all the other locations, for the boy had been circumcised in early childhood.]

Exudate from Conjunctivæ. This was flaky, and did not take the form of a uniform membranous deposit, as in the other locations involved. The same organism was found (pure) as in the other specimens, though less numerously, probably due to the active and continued treatment which the eyes had received (frequent irrigation, etc.).

Feces. Nothing characteristic was found except the same diplococci found elsewhere. The single specimen examined contained much mucus, but no fibrinous shreds.

Cultures. Pure cultures of the pneumococcus were obtained from the exudate from the eyes and nose, and mixed cultures of the pneumococcus and the staphylococcus pyogenes aureus from the membrane from the mouth and from the sputa. The diplococcus did not grow upon potato, and did not liquefy gelatin. It grew upon blood-serum, agar, glycerin-agar, and bouillon. It coagulated litmus milk, with the production of an acid reaction.

Inoculation. One minim of a suspension of the nasal exudate in normal saline solution was injected into the peritoneal cavity of a rabbit, which died in twenty-two hours with general peritonitis and septicæmia. The pneumococcus was obtained in pure culture from the heart's blood, organs, and peritoneal exudate. Stained smears from the heart's blood showed the pneumococcus typically in pairs, occasionally in short chains, with a wide capsule surrounding.

Urine. Several urinalyses were made by Dr. G. A. Himmelsbach, showing a trace of albumin, without casts, and the general characteristics of an acute febrile urine.

The eyes were practically clear and free from the exudate by January 28th. The mouth, throat, nose, and anus at this time showed improvement, but the exudate was still present in these locations in considerable amount. The membrane on the glans penis was less resistant and tended to dry up and disappear after a few days with active treatment. Gradually, after another week or ten days, the deposit over the other locations disappeared, after persisting for about four weeks in spite of active local treatment with various antiseptics. The tympanites lasted about two weeks and was a prominent feature of the disease.

The termination of the pneumonic process was by gradual lysis, as shown by the accompanying chart. The boy was allowed to sit up for the first time about the middle of February, and convalesced slowly. Slight dulness with pleural crepitation persisted over the left base, and was present when the boy was last examined, April 20, 1901.

Little remains to be said of this case after the recitation of the facts. That the production of a pseudomembranous exudate upon the various mucous membranes was caused by a specific local action of the pneumococcus cannot be doubted in view of the bacteriological findings. That the pneumococcus is capable of causing such lesions is attested by the reports of various observers. This case appears to be unique, however, in the wide extent of the process. Not only were involved nearly all the mucous membranes of the body that could be inspected, but probably the gastro-intestinal tract as well, as evidenced by the prolonged tympanites and the passage of membranous shreds and mucus in the stools. No other reported case has shown such a wide distribution. It is noteworthy also that the process was not entirely confined to the true mucous membranes, as the glans penis had been circumcised in early childhood. The infection seemed to have been transplanted from one place to another by the boy's fingers, which were bloodstained from picking at his nose and lips. The pleuræ of both sides were apparently involved in an extensive fibrinous pleuritis, and small fibrinous casts of the finer tubules were found in the sputa. All of these phenomena occurred as complications in the course of acute lobar pneumonia. There were fortunately no signs of endocarditis or pericarditis.

As but few cases of pneumococcic pseudomembranous inflammation of the mucous membranes are recorded in the literature, and most of these are of recent date, it will probably add to the interest and instruction of our case to briefly review the cases that we have been able to collect from the literature by a rather diligent search. Most of the text-books of medicine make no reference to such a process. The cases may be divided as those occurring in association with pneumonia and those occurring as a purely local infection independent of a pneumonic process, as follows:

Cases of Pseudomembranous Inflammation of Mucous Membranes Occurring in Association with Pneumonia. This class includes (*a*) cases in which the pneumococcic origin of the croupous exudate upon the mucous membranes was not proved, but merely suggested by its association with pneumonia, and (*b*) cases in which the pneumococcus was shown by bacteriological methods to be the specific agent in producing the lesion.

(*a*) *Pneumococcus not Found.* The earliest reference to croupous inflammation of mucous membranes as a complication of pneumonia that we have found is the statement by Bristowe,[1] in 1879, that "sometimes membranous patches on the mucous surface of the large intestine" occurred in acute pneumonia.

Osler[2] was the next (1885) to point out the same condition, reporting five cases of "croupous colitis" in one hundred autopsies on subjects dead of pneumonia. The process was represented usually by a "thin, flaky exudation involving only the surface of the mucous membrane. In none of the cases was there ulceration." "In one case (Case III.)

the cæcum was covered with a thin layer of adherent lymph, and scattered throughout the colon and sigmoid flexure there were numerous elevated patches of lymph, about the size and shape of rupia-crusts, which on section were found firmly attached to the mucosa. In this instance the process was very extensive, and the patches were thicker than in any subsequent case." In another case in this series of one hundred autopsies (Case IV.) "the stomach and duodenum were found greatly distended with gas. The mucosa was pale, except about the fundus, where, just to the left of the cardia, there was an extensive area of croupous inflammation, represented by a thick, adherent, grayish-white exudate, covering an area 12 by 8 cm. Beneath the mucosa the membrane was deeply injected."

Massalongo[3] is also credited with having reported a case of pseudomembranous enteritis as a complication of pneumonia.

Bardon[4] reported the case of a man, aged twenty-four years, who developed in the stage of resolution of pneumonia a pseudomembranous angina involving the entire pharynx.

(*b*) *Pneumococcus Found.* Weichselbaum[5] mentions briefly a case of lobar pneumonia and pleurisy with croupous enteritis. The pneumococcus was found in the intestinal croupous exudate, and was demonstrated by inoculation in animals.

Netter[6] observed a boy, aged three years, with varicella and urgent symptoms of laryngitis requiring tracheotomy, through the wound of which a false membrane was expelled, containing no diphtheria bacilli, but "only pneumococci with beautiful capsules and of average virulence." After some days bronchopneumonia of both bases supervened; recovery.

Rochon[7] described the following interesting case: A boy, aged two years, in the course of an attack of whooping-cough, developed severe and continued diarrhœa, with an eruption of vesicles about the anus. The examination of the clear contents of the vesicles, obtained with aseptic precautions, showed "pneumococci in pure culture" in smear preparations. During a severe fit of coughing on the eighth day of the diarrhœa a prolapsus of the rectum occurred, revealing the mucous membrane covered in places with a pseudomembranous exudate, which was removed with difficulty, leaving a red, granular base. The exudate, stained by Gram, showed "numerous pneumococci." Pneumonia of the upper portion of the left lung developed on the tenth day of the diarrhœal process, and death followed on the next day.

A. and V. Vedel[8] described the case of a child, aged two years, with bronchopneumonia at the left base and extensive pseudomembranous deposit involving the entire pharynx and the palate, lips, and nasal cavities; death after seven days from heart failure. In the pseudomembrane the pneumococcus was found in a state of purity, grown in pure culture, and proved fatal to a white rat after injection.

Ballay and Halipré[9] reported the case of a child, aged thirteen months, with bronchopneumonia, croupous exudate on both tonsils, and symptoms of laryngeal stenosis, supposed to be croupous, requiring intubation. Cultures from the tonsillar pseudomembrane showed only the pneumococcus. On the twelfth day purulent otitis media developed, but no bacteriological examination of the discharge from the ear was made. Convalescence was established on the twenty-fourth day.

Comby[10] says he has seen pneumococcic angina in two cases in infants precede pneumonia, which at first had the appearance of diphtheria.

Griffon[11] observed in a butcher, twenty-nine years old, in the course of acute pneumonia, a pseudomembranous inflammation involving the buccal mucous membrane opposite the molars on the left side, both tonsils, the palate, and the pharynx in an extensive network. Pure cultures of the pneumococcus were obtained from the pseudomembrane, and proved fatal to a mouse after injection. The croupous exudate disappeared in a few days. Tympanites was noted. A specific agglutinating reaction was claimed to have been obtained on cultures of the pneumococcus with the patient's blood.

Cases of Pseudomembranous Inflammation of Mucous Membranes of Pneumococcic Origin Independent of Pneumonia. Jaccoud,[12] who claims priority (1891) in establishing the existence of pseudomembranous angina caused by the pneumococcus, reported the case of a young man, nineteen years of age, who was suddenly taken with chill and fever, and showed on the second day a pseudomembranous exudation on both tonsils, the anterior pillars of the fauces, and the uvula. The false membrane showed all the histological characteristics of a true diphtheritic membrane, except the presence of the Klebs-Loeffler bacillus. Ménétrier, who made the bacteriological examinations, found "pneumococci in great abundance and almost pure" in the exudate, and cultures and inoculation experiments made by him "leave no doubt in this regard," *i. e.*, the pneumococcic origin of the process. The pseudomembranous process had ceased its activity by the ninth day, and the patient was well on the seventeenth day of the disease.

Jaccoud[13] also reported two similar cases in 1893, one a woman, aged twenty-six years, the other a girl of seventeen years, both with pseudomembranous inflammation of the tonsils, which made its appearance on the second day of the illness. In both cases Martin found "pneumococci in abundance in a state of purity, or sometimes accompanied by streptococci." The onset of the disease was marked by chill, shaking, and abrupt rise of temperature. Both cases terminated with recovery in three or four days. Based upon his three cases, Jaccoud attempted to draw a differential picture of pneumococcic angina, characterized by sudden onset, with chill, shaking, abrupt rise of temperature, short duration, and favorable course.

Seuvre[14] observed a boy, eight years old, who, in the course of an attack of grippe, developed an erythematous angina with signs of laryngeal stenosis, requiring tracheotomy, through the wound of which a false membrane was expelled, giving a pure culture of pneumococcus. Diphtheria bacilli were absent.

Comba[15] reported a case of pseudomembranous tonsillitis and double pseudomembranous conjunctivitis in a child of seven months, due to the pneumococcus.

Abel[16] reported a pseudomembranous inflammation in a boy of thirteen years, limited to the anterior nares, which finally disappeared after fourteen days. The deposit showed pneumococci in great abundance and a very few other organisms, but no diphtheria bacilli. Cultures and animal experiments showed that the pneumococcus was the only pathogenic organism in the exudate.

The earliest reference to pseudomembranous conjunctivitis ascribed to the pneumococcus is in the thesis by Morax,[17] who reported four cases from Parinaud's clinic, observed in the autumn of 1893, in infants ranging from eight days to two and one-half years in age. The process was characterized by the presence of pseudomembranous exudation on the lower lids, in which the pneumococcus was found pure and in abundance. The organism was grown in pure culture and recovered from animals dead after injection. The affection was always unilateral, running a short and benign course. The bulbar conjunctiva was never involved in the deposit.

Parinaud[18] reported cases of lachrymal conjunctivitis in the newborn in the first few days of life, showing fibrinous deposits or flakes mixed with the tears in the inferior cul-de-sac. In twelve cases the pneumococcus was found in abundance in the conjunctival secretion and grown in pure culture by Morax. The gonococcus and Weeks bacillus were totally absent. "This pneumococcic conjunctivitis of the newborn, as well as that of older infants, which Morax and I have described, may become pseudomembranous without ceasing to be benign, at least according to my observation."

Gifford[19] reported cases of pure pneumococcus conjunctivitis, and stated that he had also observed in these cases, as previously pointed out by Gasparini,[20] a fine pellicle of fibrin which could be wiped off from the everted upper tarsus, which had not been observed in conjunctivitis due to the Weeks bacillus.

Axenfeld[21] encountered a school epidemic of conjunctivitis, which involved twenty-four persons, showing pseudomembranous flakes. The cover-glass specimens, as well as cultures from the pseudomembranous exudate, showed characteristic pneumococci, and they were confirmed as such by Professor Behring. Animal inoculations resulted in typical pneumococcus septicæmia. The process ran its course in from three to ten days. Small ocular subconjunctival hemorrhages were noted.

As will have been observed, most of these reported cases of pneumococcus conjunctivitis were of a character that could hardly be described, strictly speaking, as pseudomembranous—*i. e.*, with an adherent fibrinous exudate on the conjunctiva—but were characterized by the presence of small fibrinous shreds in the exudate of the eye, sometimes lightly adherent to the mucous membrane of the lids. As a similar condition may often be found in the eye in almost any kind of acute conjunctivitis, it seems impracticable to draw any sharp and fast distinction between such cases and those that are more positively of a pseudomembranous nature. Within the past five years rather numerous cases[22] of pneumococcus conjunctivitis have been reported, but they have generally been without any definite pseudomembranous deposit. Such was the condition, also, in the case which we have described. The explanation of the relative freedom of the eye from croupous deposits is doubtless found in the mechanical difficulties offered to the development of such a process by the natural irrigation and drainage of the eye.

In addition to the above cited cases, which were reported more or less circumstantially, occasional references are also found in the literature to possible cases of true pneumococcic pseudomembranous angina, in which the pneumococcus is merely mentioned as occasionally found in or grown from the croupous exudate in a small percentage of a long series of cases of croupous angina studied bacteriologically, as, for instance, in the reports by Carl Janson,[23] De Blasi and Russo-Travali,[24] Cassedebat,[25] and others. The mere mention of finding pneumococci in such cases cannot, of course, be accepted as proof of the etiological relation of this organism to the morbid process, for it is well-known that occasional pneumococci may be found in the throats of a considerable proportion of healthy persons, usually estimated at 20 per cent., and can be grown in culture from such throats. Therefore, unless the pneumococcus be found in pure culture, or else as the predominating organism in the exudate, its presence is of not much significance.

The above cases, in which the pneumococcus was demonstrated as the cause of a local croupous inflammation, have all been reported within the last dozen years, and it is thus shown how recent is the recognition of the pneumococcus as an agent capable of exciting localized pseudomembranous inflammation of the mucous membranes of different parts of the body. Doubtless it is not so rare an agent in this condition as might be supposed from the paucity of reported cases, and will be shown by future investigation to be a not uncommon cause of pseudomembranous inflammation of the mucous membranes outside of the lungs.

There is nothing to distinguish the nature of the process from the similar condition associated with the diphtheria bacillus or the streptococcus, except the bacteriological examination, which should be made in all cases of pseudomembranous inflammation. The clinical course described by Jaccoud,[26] Weinberg,[27] and Gaultier[28] as characteristic of pneumococcus croupous angina[*] had no counterpart in our case, and would be of no value for excluding diphtheria, which may show the same clinical picture. As a rule, the cases have been mild, and after a short course have ended in recovery. How refractory to treatment, how long continued, and how infectious the process may exceptionally be are illustrated by our case. Most cases occur in childhood, though adults in the prime of life are sometimes affected. The mucous membranes of almost any part of the body may be involved, as illustrated by the cases cited. The process may be very limited or very extensive and almost general. It may occur in association with pneumonia during some part of its course, or may be quite independent of any pulmonary involvement. Local germicidal agents have usually been employed with apparent success, but failed entirely to have any good effect in our case. Whether due to it, or independently of it, and merely coincidently with its use—we would not venture an opinion—the process tended rather promptly to ameliorate and disappear upon the administration by mouth of drachm doses of fresh brewer's yeast.

In conclusion, we take pleasure in acknowledging our indebtedness to Professor William H. Welch, of Baltimore, for his very generous assistance in referring us to cases in the literature.

BIBLIOGRAPHY.

1. Bristowe. Theory and Practice of Medicine, second American edition, Philadelphia, 1879; and chapter on Enteritis, in Diseases of the Intestines and Peritoneum, New York, 1879, p. 11.

2. Osler. Notes on the Morbid Anatomy of Pneumonia. Transactions Pathological Society of Philadelphia, 1885, vol. xii. pp. 188–196. Philadelphia, 1886.

3. Massalongo. Mentioned in Baumgarten's Jahresbericht, 1896, vol. xii. p. 84. (Reference to original not cited.)

4. Bardon. Angines à pneumocoques. Journ. de méd. et de chir. prat., Paris, 1891, vol. lxii. pp. 479–480.

5. Weichselbaum, A. Wien. klin. Wochenschr., 1890, vol. iii. p. 187.

6. Netter. Bull. et mém. de la soc. méd des hôpit. de Paris, 1891, vol. viii., third series, pp. 224–226.

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[*] We have limited ourselves in this paper to a discussion of pneumococcic inflammation resulting in pseudomembranous exudation. It might be remarked, however, that the pneumococcus has been demonstrated as the cause of inflammation of the mucous membranes of all the following varieties: erythematous, follicular, herpetic, pseudomembranous, and suppurative. For a comprehensive discussion of these different varieties of pneumococcic angina and cases from the literature up to 1896, see Gaultier's thesis (loc. cit). In view of the occurrence of a herpetic or vesicular form of inflammation and the discovery of the pneumococcus pure in the contents of the vesicles, both in the throat and about the anus, it would be interesting to make observations on the contents of the herpes labialis, so commonly seen in pneumonia. It appears likely that these vesicles are caused by a local development of pneumococci, which would be found in the contents of the vesicles. We regret that such examination was not made in our case, and that we did not also examine the contents of the large vesicles that developed upon the chest, though in the latter case we, naturally enough, assumed the eruption to be merely the result of the local irritation of the turpentine that had been applied.

7. Rochon. Pneumococcie intestinale. Journal de clinique et de thérapeutique infantiles, Paris, October 15, 1896, pp. 843-845.

8. Vedel, A. and V. Angines à pneumocoques. Nouveau Montpellier médicale. 1898, vol. vii, pp. 621-627 and 670-676.

9. Ballay and Halipré. Infection à pneumocoques chez l'enfant. La Normandie médicale, Rouen, 1898, vol. xiii. pp. 201-207.

10. Comby. Traité des maladies de l'enfance, Paris, 1898, vol. iv. p. 86.

11. Griffon. Stomatite te angine pseudomembraneuses à pneumocoques au cours d'une pneumopathie avec état typhoïde reconnue pneumococcique de bonne heure, grâce au sérodiagnostic. Revue de méd., Paris, 1899, vol. xix. pp. 981-988.

12. Jaccoud. Angine pseudomembraneuse à pneumocoques. Journ. de méd. et de chir. prat., Paris, 1891, vol. lxii. pp. 165-168.

13. Jaccoud. Les angines pseudomembraneuses à pneumocoques. La Semaine médicale, 1893, vol. xiii. pp. 346-347.

14. Seuvre. Abst. in Revue mensuelle des maladies de l'enfance, Paris, 1898, vol. xvi. pp. 157-158.

15. Comba. Congiuntivite ed angina pseudomembranose da diplococco di Fraenkel, osservate in un lattante. La settimana medica, No. 26, 1899; abst. in Baumgarten's Jahresbericht, 1899, vol. xv. p. 67.

16. Abel, R. Zur aetiologie der rhinitis fibrinosa. Centralbl. f. bak. u. paras., 1892, vol. xii. pp. 841-845.

17. Morax, V. Recherches bactériologiques sur l'étiologie des conjonctivites aiguës et sur l'asepsie dans la chirurgie oculaire. Thèse de Paris, 1894.

18. Parinaud. Conjonctivite lacrymale à pneumocoques des nouveau-nés. Annales d'oculistique, Paris, December, 1894, vol. cxii. pp. 369-373.

19. Gifford. The pneumococcus of Fraenkel as a frequent cause of acute catarrhal conjunctivitis. Archives of Ophthalmology, New York, 1896, vol. xxv. pp. 314-324.

20. Gasparini. Annali di Ottalmologia, vol. xxiii. fasc. 6; vol. xxiv., supplement; vol. xxv. fasc. 1. Cited by Gifford, loc. cit.

21. Axenfeld. Beiträge zur aetiologie der bindehautentzündungen. Bericht über die versammlung der ophthalmologischen gesellschaft, 1896, vol. xxv. pp. 140-152. Wiesbaden, 1897.

22. Coppez, H. La Clinique Ophthalmologique, 1897.

Junius. Zeitschr. f. Augenheilkunde, Berlin, 1899, Band i, Heft 1, S. 43.

Hauenschild, W. Ophthalm. Sektion der 71. Versamml. deutsch. Naturf. und Aerzte, 1899. Abstr. in Zeitschr. f. Augenheilkunde, 1900, Band iii., Heft 6, S. 532.

Bach u. Neumann. Arch. f. Augenheilkunde, Band xxxvii. S. 57 u. 93; also Zeitschr. f. Augenheilkunde, 1899, Band i., Heft 6, S. 562.

Halle, J. Annales d'Oculistique, March, 1900.

Lundsgaard, K. Thèse, Copenhague, February, 1900. Abstr. in Archives d'Ophthalmologie, Paris, August, 1900, p. 455.

Denig, R. Zeitschr. f. Augenheilkunde, September, 1900, Band iv., Heft 3, S. 213.

23. Janson, Carl. Centralbl. f. bak. u. paras., 1893, vol. xiv. p. 143.

24. De Blasi and Russo-Travali. Riforma medica, Napoli, 1896, Nos. 179 and 180. Cited in Sajous' Annual, 1900, vol. ii. p. 565.

25. Cassedebat. Archives gén. de méd., Paris, 1897, p. 885.

26. Jaccoud. Loc. cit.

27. Weinberg, S. De l'angine à pneumocoques. Thèse de Paris, 1895.

28. Gaultier, E. De la pneumococcie pharyngée. Thèse de Paris, 1896.

ACUTE SPLENIC MILIARY TUBERCULOSIS.[1]

BY D. D. STEWART, M.D.,

OF PHILADELPHIA.

THAT the tubercular process, whether in an acute miliary or more chronic form, may have its only outward expression in the spleen, and remain there entirely limited for a greater or less period, is a fact little

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[1] Read at the Sixteenth Annual Meeting of the Association of American Physicians, held at Washington, D. C., April 30 to May 2, 1901.